

Chapter 33

Menstrual Cycle Variation in Food Intake

Douglas G. Kahn

INTRODUCTION

Variations in a number of human behaviors during the menstrual cycle are well known. The luteal phase, compared with the follicular phase, has a well-documented increase in the incidence of depression, accidents, hospital admission, suicidal thinking, and suicide. The immediate premenstrual and menstrual period in women is associated with an increased frequency of examination failure, work absenteeism, development of acute psychiatric symptoms, commission of crimes, accidents, attempted suicide, and death by accident or suicide [1,2].

The effect of food intake and body weight, lean mass/fat ratio and emotional stress on the menstrual cycle is well known (for example, the suppression of menstruation with anorexia nervosa or other starvation) [3-9]. Less well known and less frequently studied is the effect of the menstrual cycle on food intake and eating behavior. In mammals studied to date, including humans, there is a significant cyclic variation in food intake and taste preference during the menstrual cycle.

This chapter reviews the literature of the past 30 to 40 years on menstrual cycle variation in food intake and eating behavior. Studies on lower mammals (hamsters, gerbils, rats, mice, pigs, goats, and sheep) as well as sub-human primates and human studies are reported. These variations are considered significant for treatment planning in patients with eating disorders and for research on mechanisms of eating behavior and taste preference.

SUBPRIMATE MAMMALS

Most studies on variation in food intake during the estrus cycle (which lasts 4 to 5 days) have been done in the laboratory rat. Since the 1940s numerous reports have demonstrated that endogenous ovarian estrogens reduce food intake and body weight in a cyclic fashion correlated with vaginal estrus, decreased body temperature, and increased activity [10-16]. Ovariectomy results in increased food intake and body weight more than in sham-operated animals and this effect is reversed by exogenous estradiol replacement [17].

Dalvit [18] summarizes the most important findings in laboratory rats as follows:

1. Estradiol appears to be the principal ovarian hormone for regulating body weight.
2. During proestrus, when estradiol is at its peak, food intake and body weight decrease.
3. During diestrus, when progesterone is high and estrogen is low, food intake and weight increase.
4. Female rats have a higher saccharin preference than males due to the stimulatory effect of ovarian hormones.
5. After ovariectomy, meal size increases, but treatment with estradiol causes a return to control level.
6. In intact female rats, treatment with progesterone causes an increase in feeding and body weight.
7. There is no change in feeding following progesterone administration to ovariectomized rats.

Coling and Herberg [19] found increased food hoarding activity after ovariectomy paralleling the in-

creased body weight. Injection of estradiol benzoate (EB) counteracted the increases in body weight and hoarding activity as did normal estrus; progesterone and testosterone were without effect. They concluded that "estrogen-dependent changes in body weight were caused, at least in part, by a lowering of the regulated level of body weight, and that circulating estrogen (was) responsible for this." They also observed the direct non-regulating restriction of food caused by endogenous estrogen in free feeding normally cycling female rats.

Sandberg et al [20] found the injection of EB in ovariectomized (OVX) rats led to an immediate but transient suppression in both food and ethanol intake, with almost identical magnitude and time course.

The mechanism by which the preovulatory estrogen increase at proestrus proves anorexic is not known, but is presumed to be, at least in part, an intensification of short-term satiation of hunger. Drewett [10] reported that anorectic effect of the estrogen peak was limited to a reduction in meal size. Meal size on the night of estrus was reduced about 40% and associated with this was a reduction in intrameal intervals—both differences were statistically significant. Because food intake was reduced by estradiol doses too low to stimulate sexual receptivity, this decrease in food intake is not caused by the correlative increase in sexual receptivity of proestrus.

Dalvit [21] cites Wurtman, who found that rats reduce total food intake and carbohydrate intake at estrus but not their protein intake, a finding replicated in OVX rats injected with estradiol. Gray and Greenwood [22] found that progesterone attenuated estrogen-induced effects on OVX rats (including decreased body weight and carcass lipid content). Mueller and Hsiao [12] reported that estrogenic regulation of body weight was the result of two mechanisms: decreased food intake (a direct effect) and modulation of anonal growth. They found that induced weight gain in OVX rats is largely independent of food intake, thus challenging the hypothesis that estradiol decreased body weight by lowering the set point for body weight. Since their OVX animals gained weight independent of changes in food intake, they postulated that estrogen also alters metabolic processes by direct action on peripheral tissues or by action on areas of the brain regulating metabolic processes in addition to its short-term feeding-related effect (explained by a model whereby circulating estrogens bind to hypothalamic receptors to decrease food intake and indirectly lower body weight).

Nance [13] suggested that estrogen modulated feeding behavior by both hypothalamic and extrahypothalamic mechanisms. He suggested that fats and proteins may modify feeding behavior independent of

the hypothalamus and supported a primary role in neural regulation of feeding by carbohydrate mechanisms. Young et al [15] found that the factor determining response to estradiol could be altered in magnitude and dissociated from body weight by feeding different diets. Rats fed high-fat diets responded more to estradiol benzoate than did rats given chow or high-dextrose diets, and these responses were not the result of increased body weight.

Bartness and Waldbillig [23] studied intact estrus cycling rats, OVX rats, OVX rats given EB, and found no variation in total caloric intake or body weight during the estrus cycle in intact cycling rats given access to an isocaloric diet triplet of fat, carbohydrate, and protein. They did find a change in diet selection: fat intake increased, while carbohydrate and protein (to a lesser extent) decreased during estrus ... the opposite selection occurred during diestrus.

HAMSTERS AND GERBILS

Morin and Fleming [24] found a similar systematic variation of food intake and body weight with the normal estrus cycle, OVX, and EB treatment in the hamster (and noted that the same has been reported in rats, guinea pigs, and mice). They found food intake and body weight were lowest when endogenous estrogen levels were elevated. They also found a greater fluctuation in body weight than could be predicted solely by the weight of food consumed (consistent with work they cited in female rats), thus supporting studies suggesting that estradiol may in part decrease body weight independent of the direct restriction in food intake. Zucker and Wade [25] reported that estradiol failed to affect eating and body weight in female hamsters.

Observing the conflict between their study and others (including that of Zucker and Wade), Morin and Fleming suggest that rats are more sensitive than hamsters to the effect of estradiol benzoate and that sub-threshold doses of estradiol benzoate may have been used in other hamster studies. They also criticized the statistical measures used in other studies.

In concurrence with the studies of Morin and Fleming, Miceli and Fleming [26] found that estrogenic effects on food intake in hamsters were the same as those described in the rat, but were limited to specific dietary constituents. Thus estradiol decreased fat consumption without a strong effect on protein intake. However, in contrast to rats, hamsters did not show cyclic ovarian-dependent variations in carbohydrate intake.

In the only available study on gerbils, Roy found that estrogen increases body weight and food intake [27].

PRIMATES

Female primates also show a cyclic variation in food intake systematically associated with ovarian changes (levels of endogenous estrogen) during the menstrual cycle [18]. A significant decrease in food intake occurred midway through the menstrual cycle (periovulatory) and during the preovulatory phase (follicular) as compared with the postovulatory (luteal) phase, in Rhesus monkeys [27,18,28] and Chacma baboons [30].

Rosenblatt [31] found that the significant decrease in amount of food consumed correlated well with the midcycle estrogen surge, and that the amount of food consumed during the luteal phase was greater than that of the earlier follicular phase. This resulted mainly from a change in meal size. The injection of estradiol into OVX primates causes a similar depression of food intake [18].

Czaja [28] studied 202 female Rhesus monkeys for completion of meals and incidence of food rejection during nonpregnant menstrual cycles and during pregnancy. He found that food rejection incidence was greatest around the expected time of ovulation and that food rejection also "rose sharply during the third through the fifth weeks of pregnancy and tapered off during the next six weeks." He observed a positive correlation between the incidence of food rejection and the levels of circulating estrogens but not of progesterone. The similarity of food rejection during early Rhesus pregnancy to feeding changes that accompany morning sickness during early human pregnancy was noted. Morning sickness in humans is more frequently reported while estrogen levels are rising and progesterone levels declining in a time course parallel to that seen in the Rhesus monkey in early pregnancy.

Bielert and Busse [30] studied the effects of ovarian hormones on the food intake and feeding of normal, captive, wild, and OVX captive Chacma baboons. They found a significant decrease in follicular phase (preovulatory) food intake versus luteal phase (postovulatory) food intake. Exogenous estradiol benzoate inhibited food intake in OVX females. No effects of progesterone were demonstrated. These effects were independent of mating activity.

Both Dalvit [18] and Czaja [28] comment on prior studies done by Gilbert and Gilman [32] in baboons, in which Gilbert and Gilman hypothesized that progesterone in the luteal phase acted as an appetite stimulant. Czaja [28] cites more recent work refuting such a progesterone effect. He notes that progesterone antagonizes estrogen effects, and that exogenous EB (but not progesterone) has a significant influence on feeding levels in OVX Rhesus monkeys.

HUMANS (NORMAL MENSTRUAL CYCLE)

The normal physiology and neuroendocrine regulation of the human menstrual cycle is well documented. The circulating levels of hormones of the hypothalamic-pituitary axis are well known during all phases of the menstrual cycle [33-39]. Only a very few studies, however, describe the variation in food intake during the menstrual cycle in humans.

Dalvit [18] studied the self-reported dietary intakes of eight human females for 60 days (two menstrual cycles). She found that food intake averaged approximately 500 calories more per day during the ten days following ovulation than during the ten days before ovulation. This variation in food intake correlates with the estrogen levels that begin to rise at menstruation, and begin to fall after ovulation.

Dalvit-McPhillips [21] published an analysis of more of her data from this same study, looking at the differences in carbohydrate, fat, and protein consumption. She found significant and consistent variation during the menstrual cycle only in carbohydrate consumption, not in fat or protein. Her data showed that women consumed more carbohydrate per day in the postovulatory phase than in the preovulatory phase. The mean preovulatory carbohydrate intake was between 51.6% and 56.4% of the mean postovulatory consumption. Noting the parallel variation in basal metabolic rates (increased postovulation, sudden drop with onset of menses, preovulatory rise), she postulated this increased carbohydrate intake may be an attempt to compensate for the change in basal metabolic rate. She also noted the well-described anorectic effect of estrogen levels in other mammalian species. She suggested that increased carbohydrate intake elevated blood glucose levels, inducing increased levels of serotonin in the brain.

Dalvit suggested that a more useful diet strategy in the treatment of eating disorders would be to increase carbohydrate and caloric consumption seven to ten days before menstruation rather than "rigidly adhere to a sub-optimal caloric level at the time when the body's physiological needs are increased" [21].

Pliner and Fleming [17] studied self-reported food intake, body weight, and sweetness preference in 34 women. Both food intake and body weight were significantly higher during the luteal phase than during the follicular phase as measured at the midpoint of each phase. The mean caloric intake in the luteal phase was approximately 223 calories greater than the mean caloric intake of the follicular phase. Sixty-six percent of their women showed an increase in food intake in the luteal phase (independent of premenstrual fluid retention); 71% showed an increase in body weight.

Weizenbaum et al [40] found that short menses females (menstrual periods less than or equal to five

days) had increased food intake during the luteal phase, whereas long menses females (menstrual period greater than five days) did not.

Abraham et al [3] reported a study of variations in self-reported nutrient intake (carbohydrate, fat, protein, and calories) during the menstrual cycle in 23 normal women over a 35-day period. They found that "the relationship between day of cycle and intake was most obvious in respect to protein." Protein intake decreased most three days before the onset of menses. Phillips and Phillips [41] severely criticized the manipulation and adjustment of data in Abraham's study, however, and felt that the statistical measures used were inadequate.

Psychosocial factors may also cause menstrual cycle variations in food preference (and thus perhaps in intake). Snow and Johnson [42] in an article about folkloric beliefs about menstruation, report that a number of women prefer to alter their diet during the menstrual cycle. Some Central and South American women and black and southern white women believe that ingesting certain foods at the time of menstruation will result in impeded blood flow, as these foods cause blood clotting and thus disease (arthritis, being "run down").

Latin American women designate foods (and medications) as "hot" or "cold" (properties unrelated to temperature, texture, or spiciness) and avoid "cold" foods and medications on the basis of cultural beliefs at different times. The avoidance of cold foods during "la dieta"—a 40-day period of postpartum restriction—may lead to vitamin deficiency during a period of nutritional stress, since "cold" foods include citrus, tomatoes, and leafy greens. Similar cultural food avoidance has been described in Haitian, Puerto Rican, and Malaga women as well as in Cubans and Caribbean Island subcultures [42,43]. No attempt was made in either of these studies to determine whether overall food intake varied as a result of these belief-stimulated alterations in food preference.

HUMANS: VARIATION WITH PREMENSTRUAL TENSION SYNDROME

Since the 1940s several investigators have reported alterations in food preference and food intake associated with premenstrual tension syndrome (PMS) [14,18,26,28,43-46].

Smith and Sauder [47] studied a group of 300 nurses by self-report questionnaire. They found an association between a craving for food and/or sweets and premenstrual feelings of depression or tension. They also found "an association between craving at specific times (ie, during menstrual periods or depression) and the occurrence of premenstrual fluid retention" and "the desire

to eat compulsively and the tendency to be depressed more frequently." Their "sweets-craving group" differed from 30% of the subjects, who developed a craving for spicy foods and tomatoes but not for sweets. Both Smith and Sauder and Dalvit [18] reviewed prior studies done by Morton, who found that 37% of the women complaining of PMS had a craving for sweets and 23% had increased appetite at this time, and by Fortin, Wittkower, and Katz, who studied 45 women, 25 of whom had premenstrual symptoms and listed a craving for sweets as one of the most frequently reported phenomena. In summary, most studies report an increased craving for sweets in association with PMS.

A brief review article in the *British Medical Journal* [2] cited excessive thirst and appetite as among the symptoms of premenstrual syndrome and noted that PMS may persist after the menopause and is unaffected by age or parity. Endicott [45] noted that "...within a group of women with premenstrual full depressive syndrome, 35% described themselves as hypersomnic and tending to overeat."

Abraham [48] divided the premenstrual syndrome into several symptom-complex subgroups. His subgroup PMT-C is characterized by "the premenstrual craving for sweets, increased appetite and indulgence in eating refined sugar followed by palpitations, fatigue, fainting spells, headaches and sometimes the shakes." He reviewed Moo's menstrual stress questionnaire, which asked about changes in eating habits. Abraham asked about increased appetite, craving for sweets, and craving for salt. He found that 35% of premenstrual tension symptom patients could be classified as Group PMT-C. Budoff [44] mentioned that premenstrual symptoms are known to include food cravings.

Hamilton et al [49] noted that changes in appetite, including food cravings or avoidance, episodes of bulimia or binge-purge cycles, and altered patterns of drug or alcohol abuse have been reported premenstrually.

Price and Giannini [46] reported a 26-year-old woman whose two-to four-day menstrual period binge eating extended over all menses for a 12-year period, with a resultant weight gain of 2 to 4 kg in each episode. Their patient reported that binge eating alleviated feelings of anxiety, cramping, and restlessness. They also cited a report by Billiard, who described a 13-year-old girl with menstruation-linked hypersomnia and hyperphagia beginning three days before the onset of menstruation.

Price and Giannini postulated an endorphin withdrawal-mediated etiology of PMS based on their finding that a decline in beta endorphin levels correlated with severity of premenstrual symptoms. In possible

support of an endogenous opiate-mediated mechanism for stress-induced premenstrual (or other) increased food intake is a study by Roland and Antelman and a correlative study by Morley and Levine. Roland and Antelman [29] induced mild nonspecific stress in the laboratory rat by mild tail pinching. Tail pinch predictably elicited the syndrome of eating, gnawing, and licking in satiated rats, with short latency and without obvious pain. These rats preferred highly palatable fluids and familiar foods. Furthermore, such stress elicited a generalized responsiveness to the environmental stimuli at hand (rat pups precipitated maternal behavior; receptive females precipitated mounting; absence of stimulus object precipitated washing, grooming, and nail pulling). Stress-induced hyperphagia and resultant obesity were consistent outcomes of tail-pinching when food was available ad libitum. The similarity between this rat behavior and stress-induced hyperphagia and obesity in humans (premenstrual, depressed, or other) is recognized. Morley and Levine [50] demonstrated the inhibition of tail-pinch-induced eating by the opiate antagonist naloxone. This inhibition of tail-pinch-induced eating was not replicated by saline or Diazepam, both of which increased the amount of food ingested.

Budoff [44] suggested that prostaglandins may play a part in the regulation of food intake (as well as body water content and body temperature). Budoff noted that several prostaglandins have affected the food intake of rats when they were given systematically, and prostaglandin E-1 had been reported to inhibit food intake when injected into specific sites in the rat hypothalamus. In sheep the prostaglandin E-1 both suppressed and stimulated food intake depending on injection site [44]. Abraham [48] suggests that the deficiency of prostaglandin and prostaglandin E-1 might be involved in the PMT-C subgroup of women with increased sweet and food cravings.

Giannini and Price, in a recent publication [56], reported finding "a relationship between caloric intake and severity of premenstrual tension symptoms. Women who reported more severe symptoms recorded higher caloric intake." They also found that caloric intake during the premenstrual period increased with age. They did not report the magnitude of the increased caloric intake. Their method was to divide their subjects into high and low intake groups according to whether they fell above or below the median level of caloric intake for the study groups. The median level was derived from the arithmetic difference between the caloric intake 10 days premenstrually and 10 days post-menstrually. Subjects with greater change tended to have more severe PMS ($\text{CHI}^2 = 5.20, p < .05$). They found no relationship between caloric intake and progression of the menstrual cycle in their study.

MENSTRUAL CYCLE VARIATION IN TASTE PERCEPTION

Generally, female laboratory rats are known to have a higher saccharin preference than male rats because of the stimulatory effects of ovarian hormones [11,18,26]. This phenomenon is more pronounced in young rats than in old rats [30]. Similar sex differences in the preference for, or consumption of, sweet solutions have been demonstrated in hamsters and in human infants [25].

Sweetness preference in adult human females during the menstrual cycle has been observed in the fasting state and before and after glucose loads. Weizenbaum et al [40] studied sucrose pleasantness ratings in 25 females and five males studied over a five-week period. They found that males and long-menses (greater than five days menstrual period) females exhibited similar patterns, and their patterns were significantly different than that of the short-menses (menstrual period less than or equal to five days) females. Short menses females rated sucrose pleasantness significantly higher overall but did not vary during phases of the menstrual cycle.

Pliner and Fleming [17] measured sucrose sweetness preference before and after a glucose load in 41 women. They found that during the luteal phase of the menstrual cycle there was a marked decrease in pleasantness ratings following the glucose load (negative alliesthesia), and no such decrease in the follicular phase. They explained this by citing evidence that following a glucose load, glucose clearance from the blood is slower during the luteal phase than during the follicular phase, and since blood glucose levels are correlated with preferences for sugar solutions, this negative alliesthesia would be expected.

Wright and Crow [37] studied 94 normal, nonobese women. They found that the affective response (pleasantness ratings) to sugar varied significantly over the course of the menstrual cycle. They found that in the luteal phase (postovulatory), but not including the immediate premenstrual period, subjects found sugar solutions significantly less pleasant than subjects tested at other phases of the cycle (menses, preovulatory, ovulatory, and premenstrual). They also found that a significant postglucose load decrease in perceived sweetness pleasantness occurred in all subjects except those in the ovulatory phase. They note that their observed luteal decrease in sugar preference corresponds to the mid-luteal progesterone peak, and the postglucose load ovulatory decrease corresponds to the high estrogen levels. They conclude that further research in taste preference, food intake, weight gain, and hormone levels in the menstrual cycle needs to be done. Their work has been supported by Aaron's study [51].

Studies of salt preference have been performed in two species. Salt preference was studied during the estrus cycle of sheep by Michell [52], who found a maximum sodium preference six days before estrus (luteal phase), which was statistically significant when compared with six days postestrus. Kumanyika and Jones [53] studied ad lib table salt use in 24 men on a fixed daily menu constant caloric diet and 13 women on a fixed daily menu but varied caloric diet (varied with cookies only). They found "a high degree of intraindividual consistency in salt use week to week and among the women, across menstrual phases." This consistency across menstrual cycle phases in women was unexpected. They caution that if variation does occur, it may only occur in women eating ad libitum and may vary with caloric intake or diet composition, which have known menstrual cycle phase variation.

Taste sensitivity to quinine and 6-N-propylthiouracil (PROP) was studied in 19 women during one or more menstrual cycles by Glanville and Kaplan [54]. They measured taste thresholds (the lowest concentrations of the compounds that could be distinguished from water) and compared three phases of the cycle: premenstrual, menstrual, and postmenstrual. They found that in the majority of women thresholds tended to be significantly lower (more sensitive) during the menstrual period.

Henkin [55] studied sensory detection acuity for taste, smell, hearing, light, touch, and two-point discrimination during the menstrual cycle in five normal women. He found that sensory detection acuity was greater for all studied senses during the follicular phase than during the luteal phase whether cycles were long (greater than 28 days) or short (less than 28 days). He posited that "the relative increase in sensory detection acuity may be related to the effects of estrogen or progesterone on either receptor, nerve, or central nervous system activity."

SUMMARY

Almost all studies in mammalian species (humans, primates, and subprimate mammals) show systematic variation in food intake, diet selection, and/or taste preference during the menstrual cycle. The empirical evidence describes a luteal phase increase in caloric consumption. Further research is indicated in humans to validate this phenomenon, and to identify alterations in taste preference and nutrient appetites (carbohydrates vs fats vs proteins) in both the normal menstrual cycle and premenstrual tension syndrome.

Dietary strategies that respect and reflect this luteal phase increase in food intake should decrease demoralization, enhance dietary compliance and thus foster success.

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